A Historic Instance of the Adams-Stokes Syndrome due to Heart-Block.

BY

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A HISTORIC INSTANCE OF THE ADAMS-STOKES SYNDROME DUE TO HEART-BLOCK.[1]

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It is not hyperbolic to designate the subject of the present paper historic. The distinguished position which the patient occupied in academic circles; the eminent place which he filled in the scientific world; the affectionate sympathy with which his illness was followed by medical friends scattered over the entire globe; the keen interest with which he analysed his own symptoms—all these considerations render it an instance of unusual value. One of us had for more than thirty years enjoyed the privilege and pleasure of intimate friendship with Sir William Gairdner, and throughout that period had never heard of any serious illness. As is, however, widely known, he was regarded as far from robust about the date of his graduation; he was indeed threatened then with some pulmonary affection—a fact which led to his spending some months in Italy with Lord and Lady Beverley at that time.

In the year 1899 Sir William Gairdner had a severe attack of influenza, and this led him to take the step which he had for some time been meditating—of resigning his Chair in Glasgow, and coming to live in Edinburgh. He had apparently recovered entirely from all the effects of the influenzic attack long before leaving the West, and at the great banquet held in his honour—the date of which was 19th June 1900—he was in his most interesting vein. When leaving Glasgow soon afterwards, he entered with zest into all the arrangements then being carried out for the settlement of his family in Edinburgh.

During the summer of that year, one of us was residing in Strathearn, and on the 20th of August, having come into Edinburgh on business, was summoned hurriedly by Dr. Wyllie to see Sir William, who had been brought to his house in Charlotte Square in an alarming condition. Upon that day, accompanied by Lady Gairdner and one of his daughters, the patient had started from Levenhall, where he was then staying, for Edinburgh. At the station where tickets were being collected it was observed that he made no effort to produce those in his possession, and it was found that he was in a condition of unconsciousness, with a degree of rigidity of the muscles. When seen along with Dr. Wyllie, he was in a condition of almost complete unconsciousness;

[1] Read before the Medico-Chirurgical Society of Edinburgh, 3rd February, 1909.

the faee was very pale; the lips were rather blue; the pulse was feeble and irregular—its rate was only 16 per minute—the heart sounds were feeble, and it was then noted that the first sound was replaeed by a systolie murmur. Under the circumstances no attempt was made at a thorough investigation of the eirculatory condition, and our energies were entirely direeted towards bringing about a recovery from the state of unconsciousness. These were fortunately suecessful, and in the afternoon Sir William was moved to a bedroom. He gradually but steadily improved; the pulse rose during the evening and night to its normal rate, and he was ablc to rejoin his family at Levenhall in the course of the following day.

Attacks of a similar eharacter, but lcss scrious dcgree, oecurred in the following Oetober, while Sir William was in London, whither he had repaired in order to receivc thc Moxon Medal of thc Royal College of Physieians. After these incidents, as after the oeeurrence in August, he apparently recovered entirely. During thc summer of 1901 he and his third son, Dr. Erie Gairdner, went to Norway, and the visit was in every respect most successful. Unfortunately, during thc month of Oetober of that year he was attacked by pain in the right thigh and leg, attended by some œdema of the foot; Dr. Chureh, who had kindly undertaken the medieal eharge of the family, was of opinion that the eausc lay in thrombosis—a view in which Dr. Wyllie and one of us entirely concurred. About this timc, and again in December 1901, somc slight attacks oecurred, but they were not of mueh importance, and during none of thesc was the actual condition of matters watched by any medieal observer. After the seizurc of Deccmber, which oeeurred during the early morning of Christmas Day, the pulsc remained for some days irregular in rhythm, and infrequent in rate, although it was seldom below 50 per minutc.

A serious attack took plaee on 6th July 1902, in St. Cuthbert's Chureh, whieh was characterised by the pulse-rate falling to 16 per minute, with unconseiousness and rigidity. On the 16th February 1903 Sir William was attaeked while in a tramway ear, and the same afternoon he was again seized by the symptoms, and fell in Prinees Street. Two days later, nevertheless, he proceeded to London, in eompany with Sir William Turner, to attend the Meeting of the General Couneil, and in the house of Dr. Guthrie Rankin he went through several attaeks; the usual pulse-rate was noted to be 40. On the 26th February he

returned to Edinburgh, and was observed to be very faint, but without any tendeney to disturbance of eonseiousness; the pulse was about 40, as it had been in London. On the 4th Mareh two attaeks oeeurred in the early morning, with uneonseiousness and spasm, partieularly of the arms; the rate of the pulse on this day was 32. On the 5th Mareh there were five seizures during the early morning, and a few oeeurred again on the 6th. After this date the pulse-rate never rose to more than 34 per minute, while it never sank below 28, and was almost invariably found to be 32. It is very interesting, further, to observe that from this time onwards there was neither any disturbanee of eonseiousness, nor any tendeney to rigidity of the muscles, exeept on one oeeasion—in July 1905—when a transient attaek of faintness with loss of eonseiousness oeeurred. For many of these faets we are indebted to the kindness of Dr. H. Maedonald Church, who was in regular attendanee upon Sir William Gairdner and his family after they settled in George Square. His careful record has been most useful in supplementing the more scattered notes made by one of us from time to time during frequent eonsultations with Dr. Chureh, at whieh Professor Wyllie also was oecasionally present.

Sir William Gairdner took a lively interest in the development of his own symptoms, and diseussed them and their possible causation with many distinguished physicians as well as some eminent physiologists. He manifested the alertness of his mind by thoroughly grasping the various eonsiderations plaeed before him, arising out of the application of our modern knowledge to the elinieal features of his own ease. He followed with the greatest attention the results of observations by the graphie method upon his eireulatory organs, and the investigation of the arterial pressure by means of modern instruments. The tracings whieh we obtained from time to time from the veins, arteries and heart were the subjeet of his eritieal analysis, and the diserepaney between the movements of the aurieles and ventricles, whieh were then beginning to oecupy the attention of scientifie medicine, excited his elinical aeumen in the highest degree. The enormous differenee existing between the systolie and the diastolic pressure in his ease—a subjeet which was then for the first time studied —led him to refleet upon the subjeet of arterial pressure, and he never wearied of diseussing the various faetors eoncerned in its maintenance.

It was extremely eharacteristie of him that he determined to

leave a record of his own impressions and observations. He began to write an account of the course of his illness in 1903, and finished it, partly by dictation, in the course of that year. As this description[1] is full of information, it is, with the entire approval of his family, introduced in this place.

. . . . . .

"Having found reason to believe that certain attacks of illness to which I became subject in the year 1900 are, if not undescribed, at least not fully described hitherto, in their relation to other forms of organic or functional disease, I have thought it expedient to leave this record of them, and of my own case generally, for the instruction of whom it may concern, it being understood that my desire is that my medical sons, and all who have been so kind as to give me advice or assistance, shall have full discretion as to any proceeding that may throw further light on my symptoms, and also as to the form of any publication of the record that may appear to be most suitable.

At the time of the first of these attacks (August 1900) I was in my 76th year, having been born on 8th November 1824. As regards my previous health generally, I think I am justified in describing it as fairly good on the whole, especially in the latter half of life, extending to 30 to 40 years out of the 76. In early manhood I was far from strong, and, without having any apparently very dangerous illness, except typhus fever in my 21st year, I had several infirmities which bore the impress to my own mind of a rather delicate constitution—one of these being habitual constipation, culminating in very numerous and some excessively severe attacks of intestinal colic, which very uncomfortable, if not dangerous, proclivity pursued me at uncertain intervals during the whole course of my medical studies, and for many years afterwards, becoming, however, less frequent and milder in degree after I had passed my 40th year. As a younger brother had died of phthisis I was not without an impression that this, also, might be one of my predispositions, and indeed my appearance to others, as well as to myself, must have been, on the whole, the reverse of robust. It is, perhaps, not without significance in relation to the present narrative that throughout youth and early manhood the rate of the pulse was almost uniformally high, often over 100, even without any cause of abnormal excitement. I can make no precise statement as to temperature, the clinical thermometer not being then in use; but my impression is that febricula, perhaps not at all considerable, was not infrequently

present, and with it a sense of being easily chilled, and also easily flushed and overheated, from early childhood onwards.[1] Still, nothing occurred that can be distinctly cited now as amounting to evidence of tubercular disease. I never had any kind of articular disease (except, perhaps, occasionally "growing pains" in adolescence, and these not severe). There was nothing at any time to raise a question as to disease of the heart, and nothing, except the delirium of typhus fever, to suggest cerebral disorder of any serious kind, during the whole course of my life.[2] Still, I am inclined now, looking back over the whole past, to say that up to the latter part of my third decade the sense of physical infirmity, and of a possibly unsound constitution portending a more or less short career, remained with me;[3] and it was not till after removing to Glasgow in 1862 that I began to think it might turn out otherwise. In 1870 I married, and have no hesitation in saying that the years passed since this event have been much more full, not only of enjoyment of life, but of positive physical health, than those of my youth and early manhood. The colics in particular, have ceased to trouble me, though the constipation has remained.

Two remarks may be made at this stage as completing, or qualifying somewhat, the preceding account of my general health from youth onwards. (1) It would not be difficult, I think, to make out, by implication, a gouty habit in my later life; but nothing has ever occurred even approaching to an acute attack of podagra or chiragra. (2) A rheumatic tendency might possibly be inferred as derived from my mother and my ancestry on the maternal side; but endocarditis (and organic disease of the heart of any kind) I do not know to have existed for three generations back, either on the father's or the mother's side, any more than regular gout. My brother John, indeed, when in his seventh decade,

[1] I suffered a great deal, even acutely, as a child from numbness of the fingers in cold weather; and as this was not allowed for by my nurses, it was the source of much real misery to me as a peculiarity that I was disposed to be somewhat ashamed of, and yet could by no means get over, besides the sheer physical suffering it involved. At school, I never could throw snowballs on this account, and up to a much later date was always what in Scotland is called "cauld-rife."

[2] A possible exception to this statement will be noted later on, in connection with the history of the attacks.

[3] This rather persistent impression of delicate health did not hinder me from working hard at my profession, but it certainly did prevent me from applying for life insurance till about my 40th year; and probably, also, with other considerations, had its effect in delaying marriage.

had *for the first time* an attack of regular gout, which indirectly proved fatal, for he had been for many years a glycosuric, and the gouty attack led up to an unusual ending in gangrene of the sole of the foot, under which, after several months, he succumbed, latterly with a form, I think, of diabetic coma. He, like my mother, passed for being "rheumatic" at various periods in his life, but never had, so far as I know, an attack of acute rheumatism or any kind of cardiac complication. This last remark applies, indeed, to the whole family history on both sides, as far as I can trace it.

The first departure from the strictly normal condition that had subsisted for years, in respect of the incidents about to be recorded, was on the 20th August 1900, at which time the family were in temporary occupation of a house at Levenhall, while the present house in George Square was being got ready for their reception. Lady Gairdner was superintending arrangements, and in consequence of this there was frequent railway transit between Levenhall and Edinburgh. On the morning in question I had left after breakfast with my wife and our youngest daughter, without any misgiving as to the integrity of my health, and nothing occurred until the collection of tickets in approaching the Waverley Station, when it was noticed that I did not respond for some little time to the collector's demand for the tickets, and no doubt was for some moments unconscious. Before the station was reached, however, I was again partially conscious, but on leaving the carriage, relapsed, and had to be conveyed into one of the rooms attached to the station, where, however, on again regaining consciousness, I gave directions to be taken along on a truck and put into a cab, and driven to Charlotte Square. I cannot positively affirm the state either of my consciousness or of my heart's action during this drive, when I was accompanied by the two ladies, but the result of it was that we were received by Dr. Wyllie, who placed me on a sofa in his drawing-room, and gave me certain stimulants, and other necessary remedies. During the 3 or 4 hours which succeeded, there must have been many attacks of unconsciousness, the nature of which was not quite obvious to myself or others at the time, but which were no doubt attended by rather extreme modifications of the pulse, and by apparently impending collapse. Dr. Gibson, as well as Dr. Wyllie, saw me during this period, which at last appeared to come to a crisis in an attack of vomiting, after which I was removed upstairs to a bedroom, and remained for 24 hours under observation before it was considered expedient to

allow me to depart. It may be mentioned incidentally that this was the only occasion during the whole course of the illness on which an attack of insensibility ended in vomiting. Of course, the minutest possible inquiries were made as to convulsion, paralysis, &c., and also as to any apparent exciting cause for these attacks, but I think I am correct in affirming that their precise nature remained unexplained; my own impression at the time leading to the view of something epileptiform, though without the usual concomitants, other than unconsciousness, of a fit of major epilepsy. I am not sure that the total number of unconscious attacks can be stated with accuracy, but they were numerous, and in all of them the heart was gravely affected, but the nature and extent of the affection was probably not very accurately noted at the time.

On recovering from this series of attacks, I resumed the ordinary course of my life, and had nothing definite to complain of for at least 8 weeks. On the 18th of October, however—St. Luke's Day—I was due to be in London to receive a clinical medal —the Moxon Medal that had been awarded to me by the Royal College of Physicians—and although the question was raised as to the propriety of my going to London, I felt so well as to leave on the 17th without misgiving, going to my club in St. James Street, where, fortunately for me, my friend, Dr. Gordon Sanders, was for the time also domiciled. On the invitation of Sir Dyce Duckworth, and after a simple and early dinner, we went down, all together, to St. Paul's to a service, in great part musical, on behalf of St. Luke's Guild, which was very largely attended, and even up to the commencement of the service I had still no misgivings, although feeling considerably fatigued by the very dilatory process of marshalling the robed guests who were in the nave in procession. As there was no insensibility on this occasion, and as I am not even now quite confident as to how far it resembled the preceding or succeeding attacks, I will only say that during the sermon I was only restrained from leaving the church by the prominence of my position, sitting immediately in front of the pulpit, and that as soon as the service was over Dr. Sanders and I made for my club in a hansom, only too glad to be relieved from a sense of impending disaster, which might have led to a scene. The succeeding night was one of great discomfort, attended by both sickness and diarrhœa, of which the only possible explanation that could be suggested as regards the dinner was that I had eaten of mushrooms. In this attack, however, there was no insensibility,

nor am I aware that the rate or character of the heart's action was at all profoundly affected. At all events, on the 18th I felt so much recovered that I not only attended at the College and received my medal, but attended the annual dinner of the College thereafter, and made a speech as a guest without any sensation of discomfort, returning home the next day apparently in my usual health.

Between the events in the autumn of 1900 and those now about to be related there intervened a period of what I should call fairly good health, in which I remember no incident at all worthy of notice in this record, unless it be that the knowledge of what had occurred may have made me more careful, both in taking ordinary exercise and in committing myself to engagements away from home. I attended the meeting of the Medical Council in the summer of 1901, staying in London at my club without any serious interruption; it was then proposed to me to make a journey to Norway with my son Eric, but this proposal was made as much on his account as on mine, he being supposed to be in need of a holiday in the interval of his medical studies. I had been in Norway more than 20 years before, but I very gladly accepted the proposal, and we sailed in the beginning of August by the s.s. *Midnight Sun* from Newcastle, the trip occupying a fortnight. During the whole time I was perfectly well, and enjoyed everything very much, but was conscious of an indisposition for much physical exertion, and I did not, therefore, attempt any of the longer excursions involving either walking or climbing, but confined myself to such as could be done by driving, with a little walking uphill. It never occurred to me, however, that I was breathless in any serious degree, even in walking up the zig-zag road from Gudvangan, which I did to the extent of several hundred feet, though with a certain sense of fatigue, but not at all of serious oppression. I feel quite confident in stating that, apart from a sense of a diminution of vigour, and, perhaps, a degree of natural apprehension of the consequences of over-exertion, I never felt a single hour's discomfort during the whole of the Norway trip, and did not lose a single night's sleep.

About this time I had some slight inconvenience amounting to pain, and even a certain amount of halting, with tenderness in the calf of the right limb, which in connection with the previous incidents led to Dr. Church and Dr. Gibson being consulted as to the possibility of some kind of thrombosis. Nothing of the kind was positively detected at any time, but the inquest into this led to the discovery of some very slight tendency to œdema, so slight, however,

that I myself was not quite convinced of its actual existence, although both my sons and Dr. Church held it to be occasionally present, and I, therefore, agreed to pursuing a more or less diuretic course of treatment, with observations on the quantity of the urine, which, however, did not lead up to any very definite result. Digitalis was commenced about this time, and although the quantity of the urine was quite satisfactory, and sometimes, indeed, though not commonly, rather in excess, the digitalis was continued in various combinations, and, especially after consultation with Dr. Balfour, it was, as a rule, part of the treatment, even although not producing any marked physiological effect.

About a fortnight after this, in November 1901, an attack occurred in the very early morning, being the first time that the seizures had taken place while in bed and while asleep. It was noticed by my wife in consequence of some disturbance of the breathing, and my son Eric was immediately sent for; he found the pulse below 40 in the minute, but pretty full and regular. It is supposed that the unconsciousness on this occasion may have lasted an hour, and it must have led to some apprehension, for two hypodermic injections of ether were administered without any sign of sensibility. There was a certain amount of cyanosis and coldness of the extremities, which were met by a local application of hot water, and hot water administered by sips internally, which were swallowed unconsciously. On the termination of the unconsciousness, the pulse returned to the normal rate.

On Christmas morning, 1901, about 7 A.M., an attack occurred somewhat like the last, but more brief, the unconsciousness lasting only 10 minutes; during this attack the pulse was found to be 36 per minute, regular, and not at any time difficult to catch; similar remedies were applied. The only reason for mentioning this attack separately was that it was followed by a period of some weeks characterised sometimes by infrequency, and sometimes by irregularity, of the pulse, though without any further unconsciousness. During the Christmas week almost every possible kind and degree of irregularity was observed, sometimes alternating full and small pulsation, sometimes 3 or 4 comparatively rapid and small beats followed by a fuller one. As these irregularities were not accurately and individually noted at the time, and as no tracings were then got, it can only be said in general terms that diminished rate did not infer irregularity, but rather the opposite. On New Year's Eve Dr. Church called and found the rate a little over 30 in the minute, but the rhythm so regular that he was

asked to compare it with the cardiac rhythm in order to make sure that the low rate felt at the wrist did not signify double the rate at the heart, with alternating smaller beats which did not reach the wrist; Dr. Church, however, satisfied himself that there was a true bradycardia, the rate at the heart exactly corresponding with that at the wrist. The subjective sensations connected with these variations were not very easy to define. Knowing, as I did, all the facts, it was not possible that I should be altogether unmoved by them, and I think it fair to say that this week was to me one of a certain amount of physical discomfort. On the other hand, there were none at all, or next to none at all, of what could be called grave cardiac symptoms, no trace of anginoid symptoms, or of pain in the cardiac region, or of dyspnœa properly so called; the nights were, as a rule, undisturbed, and, if there was any relative insomnia, it was only from a certain amount of apprehension, founded upon the pulse, that one of the attacks already mentioned might possibly be impending. In carefully studying, as I did, the degree to which I had any consciousness of these irregularities of cardiac rhythm, I found that in certain positions when in bed I could count the cardiac pulsations without feeling for them either at the heart or wrist, but in other positions, and for much longer periods, I was practically unconscious of the cardiac pulsation, and, on the whole, I find it difficult to determine whether the subjective state above referred to can be called morbid or not: it certainly did not *per se* amount to a serious inconvenience, and did not cause me much anxiety, or interfere practically with a single night's good rest.

On the first Sunday of July 1902 I went as usual to St. Cuthbert's Church, being at the time, according to my own reckoning, and subject to the previous statements, altogether without misgivings, and as nearly as may be in perfect health. I had, however, to a certain extent, the habit of sparing myself, and not infrequently avoided standing up during the singing for at least a part of the service. On this occasion all went well until near the end of the sermon, and then for a few seconds (no one else observing the fact) I became aware that I had passed into an unconsciousness which was not sleep, but was attended with something of the disagreeable sensation of former attacks. There was a baptism of a number of children to follow, and being very much interested in this part of the service, I stood up, though quite realising that this was perhaps imprudent under the circumstances, and noticing also that the atmosphere of the church was somewhat

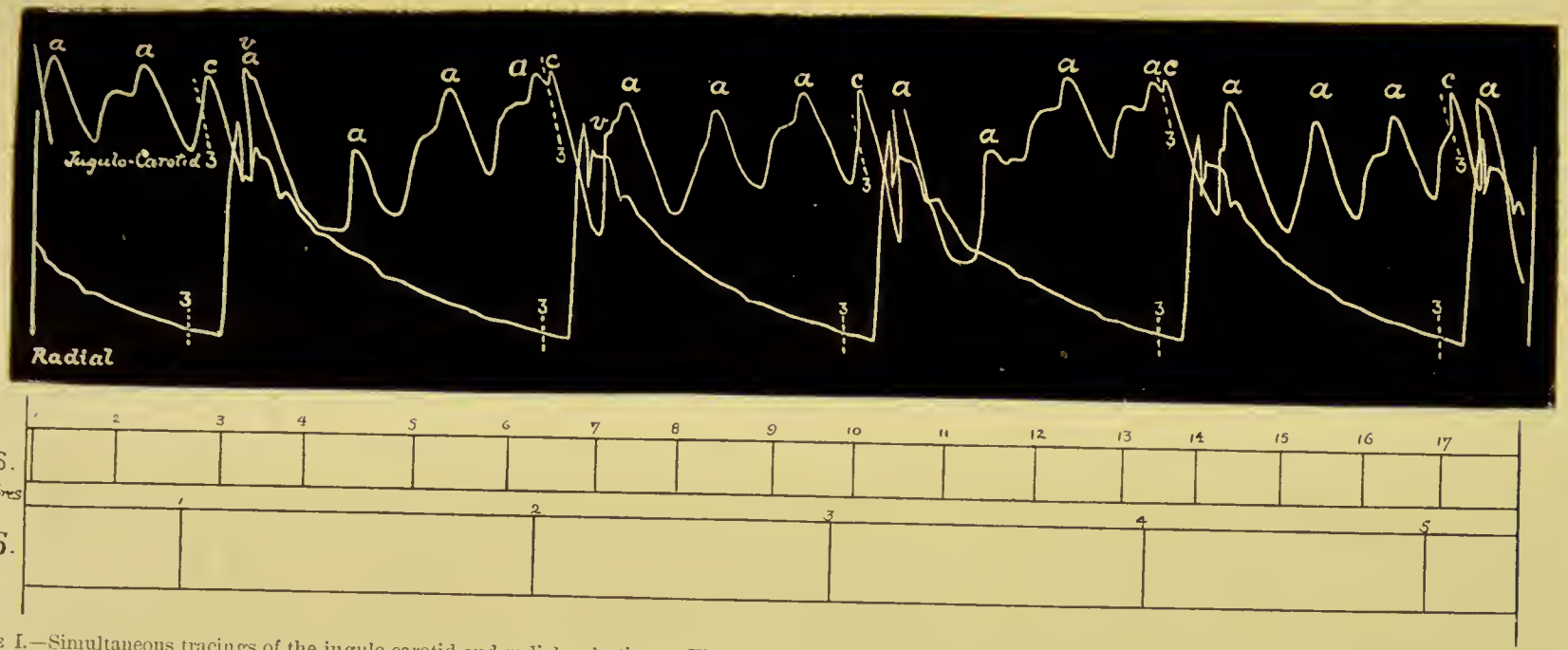


PLATE I.—Simultaneous tracings of the jugulo-carotid and radial pulsations. The diagram, constructed from the events recorded in the tracings, shows that no stimulus passes along the *a-v* fibres to the ventricles, but that there is complete dissociation of the ventricular rhythm (*V. S.*) from the auricular (*A. S.*).

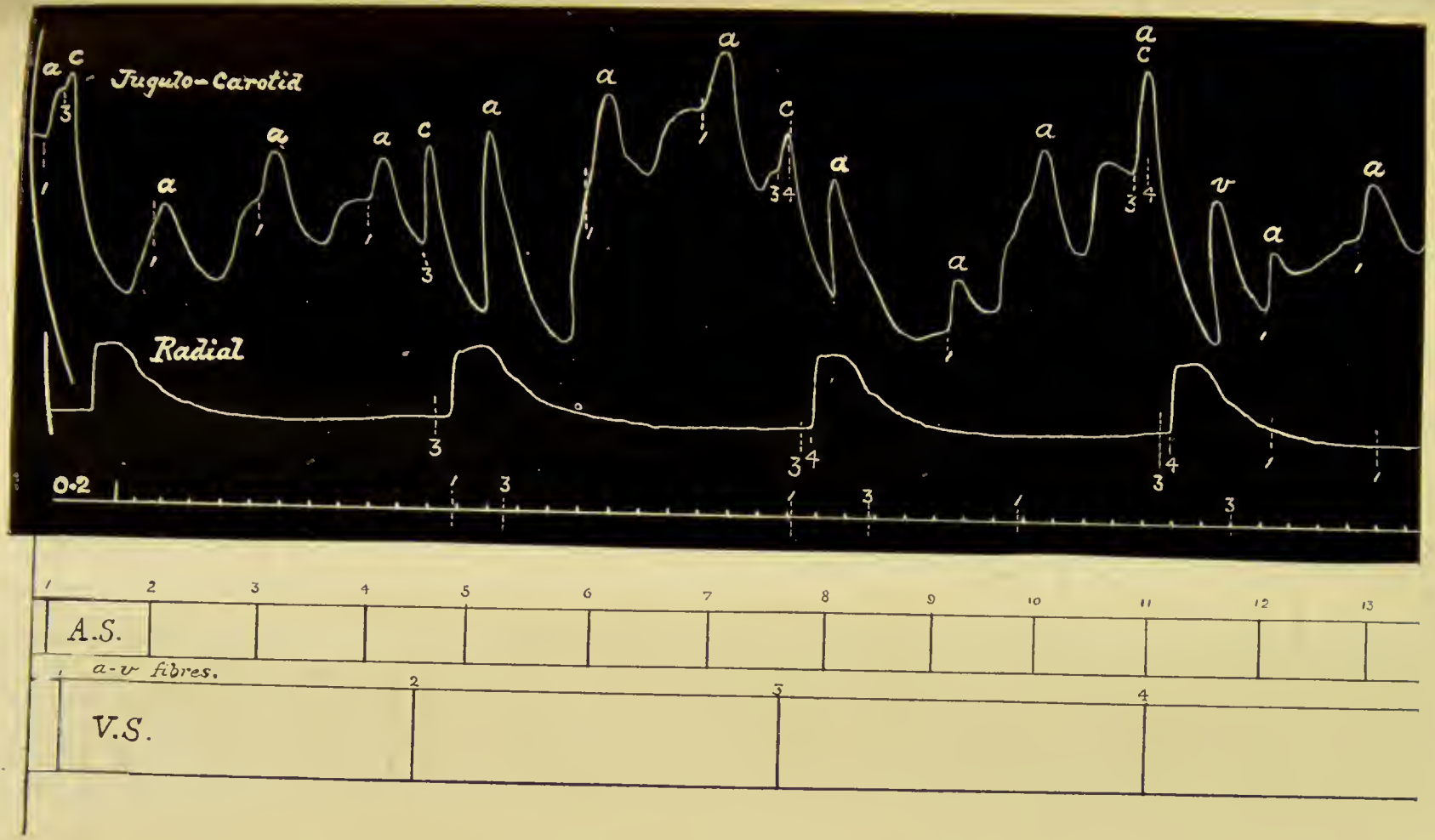
oppressive. Perhaps as a consequence of this, another attack occurred, which attracted attention, and I had to be carried out of the church into the vestry, where I soon became sensible again, and found that Dr. Carruthers was attending, and administering some whisky, as I have no doubt he had recognised symptoms of cardiac failure. I was soon, however, in a condition to be taken home in a cab, and was then placed in bed, and received all the attentions that were considered necessary. For several hours following this attack of unconsciousness I think it must be inferred, from all that came to my knowledge, that my life was in considerable danger; the pulse, which had been very small since the attack, being numbered as low as 16, and never above 30 per minute, while the face was bedewed with cold perspiration, and the subjective sensations were certainly those of dissolution at various times, but still nothing like cardiac anguish, or considerably disordered respiration. In fact, I can say with complete conviction that, although my state, medically considered, must have been dangerous, and though I was fully conscious of the danger, I had at no time any degree of physical distress or pain corresponding with this, and was perfectly free from anxiety, moral or physical, except such as is inseparable from the idea, several times expressed in words, of possibly, or probably, parting from this life, and all its interests and cares, within a space measured by hours or minutes, as the case might be. When two of my children came to tell me they were going to church in the evening, I took leave of them with the inward feeling that I might not see them on their return;[1] but notwithstanding this, I had no sense of depression, or of overpowering emotion, and certainly no such fear or sense of instant death as to correspond with what I have myself written about as *angina sine dolore*. The clammy cold sweat, however, continued, and the sensations, though certainly not agreeable, were not painful, and not in any definite way to be referred to the heart, except by reasoning from the state of the pulse, which continued up to about 7 P.M. (or over 6 hours from the attack in church) to number not much over 30 per minute. At this time another attack of unconsciousness occurred, and my son, who was with me during the whole time, reports that this was, in his view, the most alarming of all, in respect that there was

[1] This remark by Sir William vividly recalls the account of the symptoms of Mr. Hyde, father of the first Lord Clarendon, *vide Life of Edward, Earl of Clarendon*, written by himself, Oxford, 1759, p. 9. The passage is quoted by Gibson, *The Nervous Affections of the Heart*, Edinburgh and London, 1904, p. 3.

considerable cyanosis, and that the respiration appeared to be suspended, the eyes at the same time staring, and not responding to the reflex of the eyeball. There never was, as my son tells me, anything of the definite eharaeter of the Cheyne-Stokes respiration. The attack just described was, however, very brief, lasting not more than a minute; the pulse continued without ehange at a little over 30 till after eonseiousness was restored, and then eame a kind of crisis, the whole of the unfavourable symptoms passing rapidly away, the pulse returning to 70 a minute, and perfeetly regular, with complete warmth and eomfort. I have dwelt on these details beeause this attaek, lasting in all about 7 hours, was by far the most serious I ever experieneed, with the possible exeeption of the first one (or rather series of attaeks) in the autumn of 1900; and although undoubtedly eardiac as regards the bradyeardia, it left on my mind (as the previous ones did) the impression that the cerebral element predominated.

After eonsiderable additional experienee of the attacks previously adverted to during the last few weeks, it is now, I think, possible to elassify the whole series oeeurring sinee the autumn of 1900 as follows:—1. In most, if not all, of the earlier attaeks, namely, those that took plaee quite unexpeetedly, and in the midst of what seemed to be good health, it now appears to me that there must have been quite suddenly a brief, sometimes momentary, loss of conseiousness, more or less eomplete, assoeiated with a praetieally simultaneous fall in the rate of the pulse of from 20 to 40 beats in the minute—the latter being always eonsiderably more persistent than the unconsciousness, and extending over from half an hour to several hours, but ending in a return to the normal rate. It is diffieult to be quite sure of the details as to the bradycardia, beeause of its having supervened so suddenly as not to have been quite closely watehed at its beginning, but in typieal instances when either I myself, or some near relative, was able to make the observation, the pulse immediately after the period of unconseiousness was found to be small, and of a rate varying from 20 to 40, but, as far as observed always quite regular at this rate, and reverting towards the normal sometimes with the oeeasional omission of a beat, or slight irregularity during the rise to the normal rate.

2. The above deseription applies probably to all the attaeks oeeurring unexpeetedly in the midst of normal health. At a later period, towards the close of 1901, when considerable irregularity of the pulse was noticed extending over several weeks, and of the


Jugulo-Carotid
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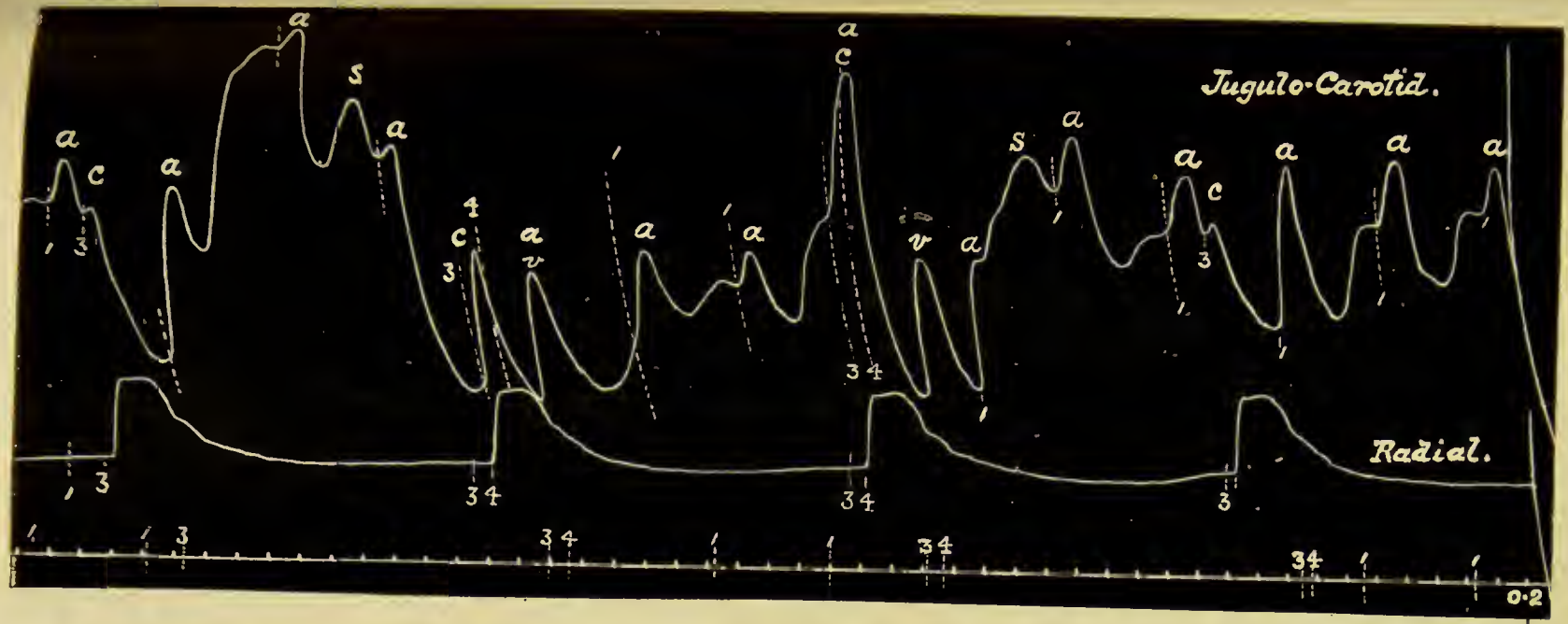
most varied possible character, bradycardia still accompanied some of the attacks which were not, however, so characteristic or so long continued in their effects as in the first series; of this period, however, no very detailed facts can be given, and, on the whole, the frequency and character of the attacks of unconsciousness bore no proportion to the irregularity of the pulse, but rather, if anything, seemed to be less prominent when the latter was at its height.

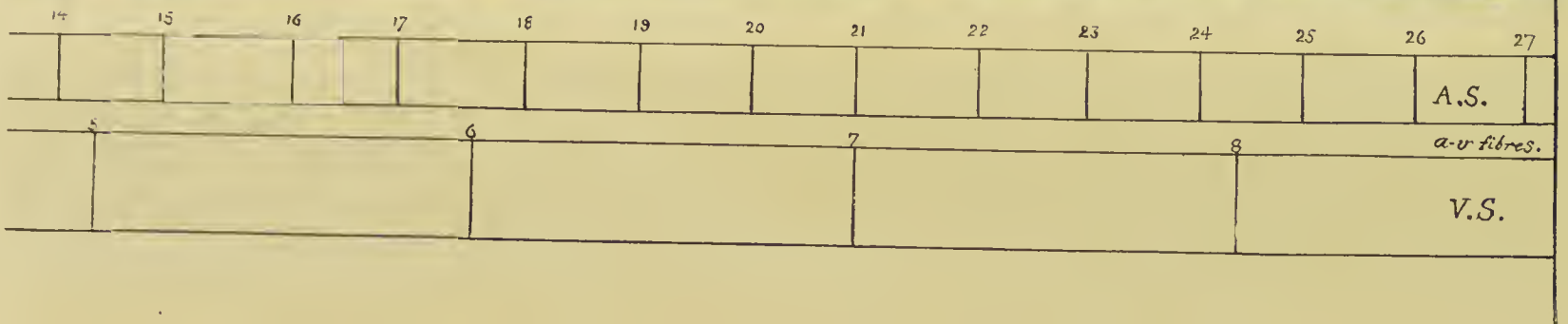
3. In the later periods of the case, and especially within the last 3 weeks, bradycardia has been present, and is still present, with a continuity not known at any previous period, the rates varying from 20 to 40, and perhaps averaging rather below than above 30, but never quite so low as after the nearly fatal attack which commenced in St. Cuthbert's Church. During this last period the attacks of unconsciousness, though mostly very brief, and measured by seconds, have been much more frequent, and in a certain sense disturbing, than they ever were before; they have mostly, though by no means exclusively, occurred at night, and often for many nights consecutively (not infrequently during sleep), and attended by sensations almost indescribable, but possibly more allied to vertigo than anything else—very molesting at the time, and possibly tending to exhaustion, but not accompanied by any cardiac sensations whatever, and even as regards the pulse, of which I was always unduly conscious, not conveying in the least degree the feeling of syncope, or of angina pectoris, but rather that of an internal throb throughout the chief vascular areas which I often watched carefully, comparing it in my mind with the dull throb which I experienced in my berth in an Atlantic liner in 1891, and which conveyed no sense whatever of panic, or even of great inconvenience, beyond the fact of its being obviously abnormal. It is notable that while during the last few days the frequent attacks of unconsciousness have entirely given way to a sense of comparative comfort, and perhaps an average of from 8 to 10 beats rise in the frequency of the pulse, which have never, however, exceeded 40, these vascular throbs have been much less apparent, and have indeed almost disappeared from the consciousness. I must again most emphatically state that, according to my later experience, the frequently recurring cerebral attacks appear to myself to have no cardiac symptomatic expression, and in most cases were not accompanied by any manifest depreciation of the rate of the pulse, but seemed, on the other hand, to be always accompanied and followed by cerebral perturbations, of which I find it quite impossible to give any description

that would convey to others a clear idea of what they were to myself. I make this remark after having carefully watched an almost indefinite number of these attacks, in some of which the unconsciousness was so slight and incomplete that I was able to realise the subjective phenomena from beginning to end. A good many of these very minor attacks were distinctly attended by what I should incline to call an aura, mostly as if a cold breath of air were playing upon the face, sometimes, however, the sensation being rather of warmth than of cold, and never spreading over the body generally. I have put the question repeatedly as to whether any obvious motor spasm had accompanied these attacks. Anyone who has been with me for some time says there was some rigidity of arm and wrist, and also a degree of risus sardonicus; these possibly belonged to the beginning of the attack, and to the period of complete unconsciousness. There was at no time any cry, but the occurrence of the seizure was often noticed through perturbations of the breathing. About 10 days ago, when my own sensations were certainly those of closely approaching death, there was still a complete absence of anything like cardiac oppression or inconvenience of any kind, the sensation being simply that of vitality ebbing away without suffering of any kind, and without anything approaching what I take to be the sensations of syncope (though I have rarely experienced them), and can only further state that the pulse, even at the worst, though with marked reduction of rate to 20 in the minute, or thereby, was never at any time markedly irregular or imperceptible to the finger. I should like also to mention that though I have had a considerable amount of flatulence and other digestive disturbances during the varied course of these complaints, it appears to me now to be quite certain, after careful watching, that in no instance was either gastric or intestinal flatulence, or any other digestive disturbance, a contributor in any marked degree to the occurrence of the attacks."

This description, founded upon the observations which the patient made upon himself at the time of the various seizures and recorded at the later period when the intermittent attacks of infrequent pulsation, with consequent fainting, were giving place to persistent reduction of pulse-rate, unattended by any nervous consequences, is certainly one of the most interesting contributions to the literature of cardiac disease.

It will be observed that there are two discrepancies between the impressions described by Sir William and the facts recorded


Jugulo-Carotid.
Radial.
0·2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by his medical attendants. In the first place, there can be no doubt that the attacks of infrequent pulse and cerebral symptoms were sometimes, at least, very definitely caused by digestive disturbances. The first seizure, for example, was undoubtedly produced by imprudence in diet. As this is a matter of practical importance, it is only right to refer to it. In the next place, he very decidedly expresses the opinion that the attacks of illness were of cerebral, not cardiac origin; he was somewhat inclined to such a view as one of the members of his father's family had suffered from epilepsy; this belief, however, he modified during the later months of 1903, and in the course of 1904, after carefully studying with us the graphic records of his heart and vessels.

Having narrated the general course of the symptoms presented by Sir William Gairdner, we have now to enter upon the results of physical investigation and instrumental examination, carried out for the most part after the development of the permanent infrequency of the pulse.

The complexion, although pale—as it had indeed been throughout his life—was healthy; in fact, during the whole of his illness there was no alteration in colour except during the worst of the attacks, when, as has already been mentioned, there was sometimes a degree of cyanosis. The general state of nutrition was excellent, and there was no trace of emaciation; the muscles retained their tone wonderfully well, considering the small amount of exercise which could be allowed; the skin remained fresh and smooth.

The tongue, as a general rule, was clean, and, apart from some flatulence, there was no complaint in regard to the alimentary system. The stomach was only moderately dilated, but the colon usually showed very considerable distension. Constipation had been a trouble throughout most of his life, and it required to be carefully guarded against during his later years. Apart from the distension of the colon, physical examination of the abdomen revealed no abnormality, and the liver was of the usual size. Examination of the blood showed no departure from normal conditions. There was no trouble in respect of the renal functions, and from first to last there was neither disturbance in the proportions of the normal constituents nor the slightest trace of albumin or of any other abnormal constituent. Careful examination of the lungs showed slight dulness on percussion over the apices, both in the supraclavicular and supraspinous regions; this was attended by a harsh respiratory murmur, scarcely, however,

amounting to bronchial breathing, and accompanied by a moderate degree of accentuation of the vocal resonance. The nervous system, carefully examined, scarcely yielded a trace of any departure from the normal standard. There were rarely any subjective sensory disturbances, and sensibility, tested objectively, was intact. Sir William had suffered for some years from detachment of the retina, and the sight was therefore considerably impaired. Hearing was somewhat dull, but up to the date of resignation he was able to auscultate perfectly, the degree of deafness which was present being due to changes in the media, and not in the nervous mechanism. The organic and cutaneous reflexes were intact, while the muscle and tendon responses were lively, but not exaggerated; the muscular system, although necessarily far from strong, was in no respect abnormal.

No vasomotor or visceral symptoms were present, excepting that during the minor attacks the face manifested a higher degree of pallor than usual, while the graver paroxysms led to some cyanosis.

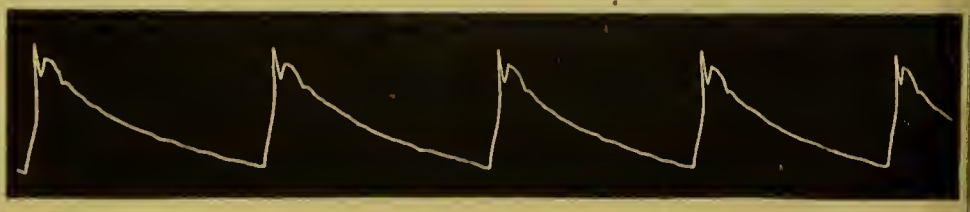
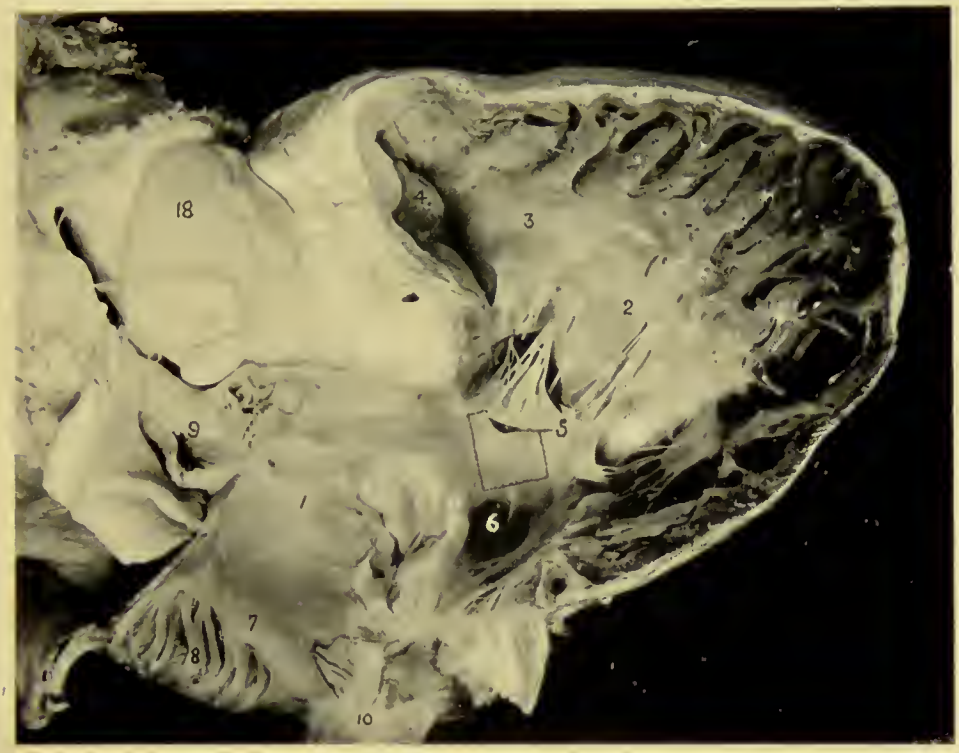


Fig. 1.—Tracing from the radial artery.

Throughout his life, as he indeed mentions in his own description of his symptoms, there was a tendency to paleness. The innervation of the viscera was in no sense interfered with. We were inclined to regard a temporary digestive disturbance as sometimes the cause of the attacks of the Adams-Stokes syndrome, which, as is now well recognised, is sometimes brought about by reflex stimulation through the vagus nerve. The intellectual processes remained to the last day of life perfectly unimpaired, and to the very end Sir William Gairdner manifested wide interest in, and acute appreciation of, every modern movement, whether scientific, literary or political.

The arteries within reach of examination by palpation were slightly thickened, but not hard; they were compressible quite easily, and showed no tendency to tortuosity. The vessels gave to the finger the sense of a large, well-sustained impulse, with comparative emptiness between the pulsations. The rate of the pulse during the last four years of life varied between 24 and 32 per minute (Fig. 1). The rhythm was not absolutely



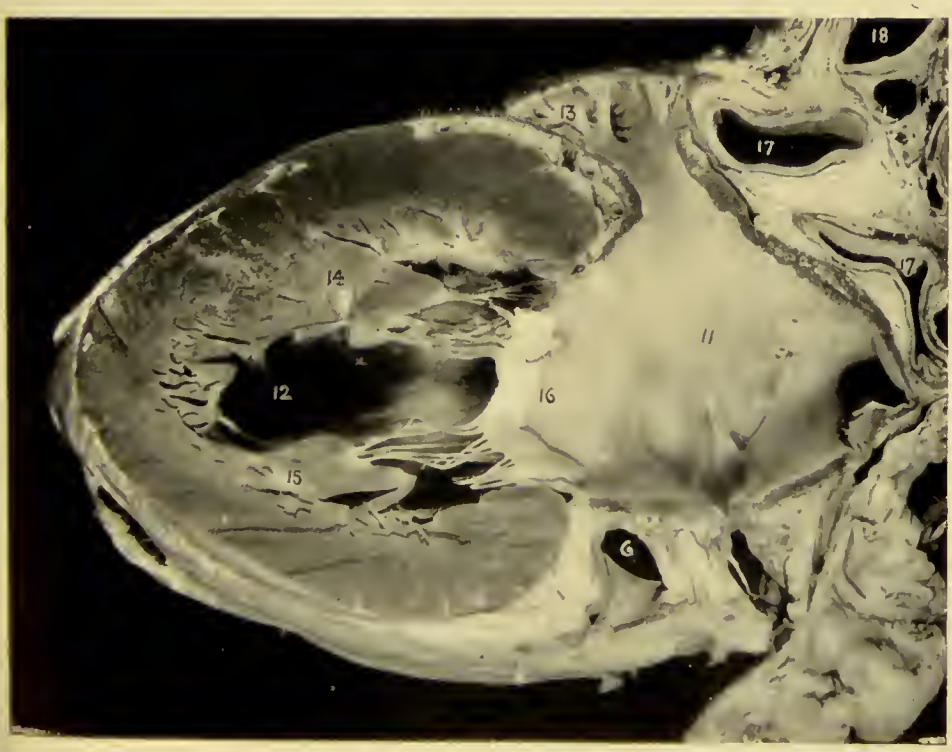


PLATE IV. FIGS. 1 and 2.

1. Septal wall of right auricle. 2. Septal wall of right ventricle. 3. Infundibulum. 4. Pulmonary valve. 5. Tricuspid valve. 6. Mouth of the coronary sinus. 7. Tænia terminalis. 8. Pectinate muscles of right auricle. 9. Superior vena cava. 10. Inferior vena cava. 11. Septal wall of left auricle. 12. Septal wall of left ventricle. 13. Left auricular appendix. 14. Anterior papillary muscle of left ventricle. 15. Posterior papillary muscle of left ventricle. 16. Aortic cusp of mitral valve. 17. Main branch of pulmonary artery. 18. Aorta.

regular, as numerous tracings showed slight variations in the length of the systole. These, however, were by no means striking. The arterial pressure, which was for the first time in such conditions carefully studied, revealed features of the greatest interest; the systolic pressure was always between 200 and 300 mm. Hg., usually between 210 and 270; the diastolic pressure only varied between 75 and 85. Unfortunately, the sphygmomanometer devised by one of us[1] was not sufficiently developed during the patient's lifetime to enable us to obtain records, but a tracing taken from the patient with Erlanger's instrument (Fig. 2) shows a systolic pressure of 230 and a diastolic of 75. In a previous communication upon heart-block[2] we have discussed these interesting facts of arterial pressure, and have attributed the enormous difference between systolic and diastolic pressure to

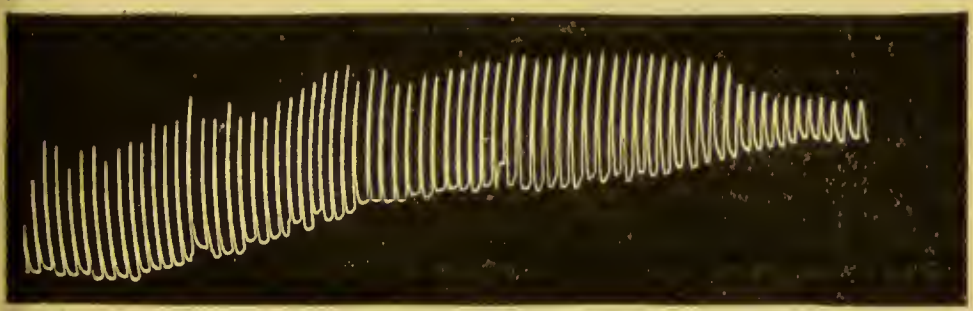


Fig. 2.—Tracing taken with Erlanger's sphygmomanometer. The systolic pressure is 280, the diastolic 75 mm. Hg.

the long interval elapsing between the contractions of the ventricles.

On inspection of the cervical region faint movements were obvious in the external jugular vein, and vigorous pulsation was seen in the carotid arteries. It was perfectly clear that two or three flickering oscillations took place in the vein between two consecutive arterial pulsations. The apex beat was vigorous but somewhat diffuse, its maximum intensity being just outside the mammary line. No abnormal pulsations could be seen in the precordia, and, on palpation, the powerful apex beat was found to be accompanied by no thrill, and no adventitious movements were communicated to the hand. On percussion, the right border of the heart, at the level of the fourth costal cartilage, was 2 inches from midsternum; the left border was just outside the mammary line

[1] *Quarterly Journal of Medicine*, 1907, vol. i. p. 103.
[2] *The Practitioner*, 1907, vol. lxxviii. p. 589.

4¼ inches from midsternum. The first sound over the whole precordia was replaced by a loud, high-pitched, blowing murmur; its maximum intensity was found to be at the apex, whence it was propagated most distinctly to the axilla and towards the angle of the scapula, as well as, although less distinctly, throughout the whole chest. The second sound was loud and ringing; its point of maximum intensity was about the aortic cartilage and the right border of the manubrium sterni. In that region there was a slight alteration of the character of the systolic murmur, which was found to be conveyed to the carotid arteries; it was therefore assumed that an aortic systolic murmur accompanied that produced at the mitral and tricuspid orifices. The condition was undoubtedly that of a senile heart with escape at the great venous orifices from stretching of the muscular sphincters, while it seemed certain that there was, in addition, some dilatation of the ascending portion of the aorta, revealed by the systolic murmur and accentuated second sound.

Another phenomenon revealed by auscultation was of the greatest interest. Following every second sound, and therefore preceding every systolic murmur, there could always be heard perfectly distinct, if not loud, sounds, usually two in number; these were most distinctly audible along the right side of the sternum and towards the axilla, where they became somewhat less distinct. On passing still further back, towards the angle of the right scapula, these sounds again became much more evident; they could be heard at the apex of the heart and along the left edge of the sternum, but they were not so evident, and it was difficult to hear them further back over the left side of the chest. These sounds were assumed to be caused by the contraction of the auricles, and on auscultating with the binaural stethoscope, they were found to precede the flickering movements of the jugular veins. These auscultatory phenomena produced by the auricles have been fully discussed by one of us in previous works,[1] and by both of us in a subsequent communication.[2] The examination of the cardiac movements by fluoroscopy, which was first introduced by one of us,[3] was never carried out in the case of Sir William Gairdner, as it was felt that in his condition of health the inevitable disturbance produced by this

[1] *The Nervous Affections of the Heart*, 1904, p. 61 ; and *Edinburgh Medical Journal*, 1905, vol. xviii., N. S., p. 9.

[2] *The Practitioner*, 1907, vol. lxxviii. p. 589.

[3] *Proceedings of the Royal Society of Edinburgh*, 1905, vol. xxv. p. 1085.

method of examination was not advisable, and this additional aid to diagnosis was not therefore adopted.

Graphic records of the arterial and venous pulsations, and of the apex beat of the heart, however, were obtained from time to time, and they afforded confirmatory proof of there being complete dissociation of rhythm of the auricles and ventricles, or, in other words, of complete auriculo-ventricular heart-block.

The tracings reproduced in Plate I. are the enlarged records of the radial and jugulo-carotid pulsations taken simultaneously in 1904 with Mackenzie's clinical polygraph. Below the tracings we have placed a diagrammatic interpretation of the events recorded. In Plates II. and III. there are reproduced the tracings, somewhat reduced in size, that were taken in 1906 by means of the Knoll-Hering polygraph; the time is recorded by Jaquet's chronograph in fifths of seconds, and the accompanying diagrams are constructed to illustrate the events recorded in the tracings.

Each auricular wave in the phlebograms is indicated, in the usual manner, by the letter *a*, whilst *c* is the carotid elevation, and *v* the ventricular wave. In the *tracings*, the numeral 1 marks the time of commencement of the auricular wave; 3 indicates the appearance of the carotid wave; and 4 the moment at which the radial pulse wave appears. In the subjacent *diagrams*, the auricular beats, and likewise the ventricular, are numbered consecutively.

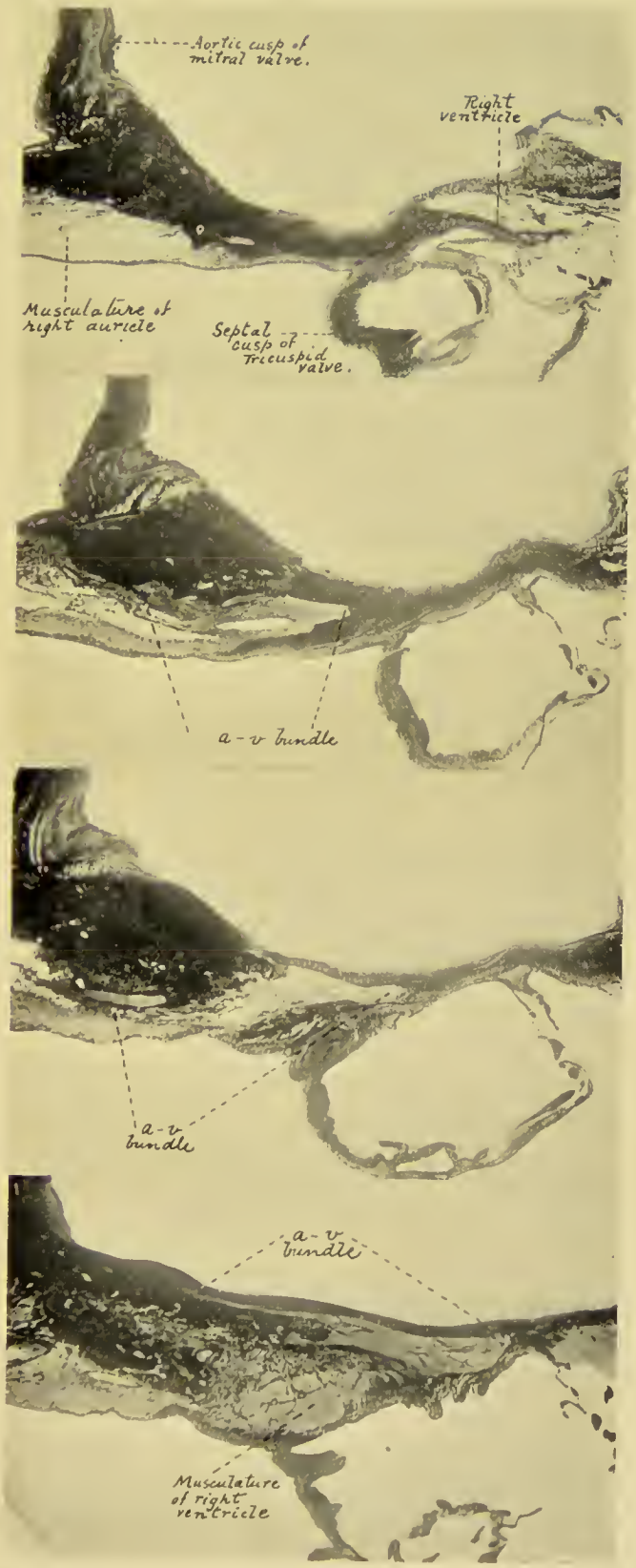
The tracings in Plate I. show that the auricular rate is more than thrice as frequent as the ventricular. In Plate II., where the time record is available, we find that the rate of auricular contraction is 80 per minute, while that of the ventricular beats is only 24·4 per minute. In Plate III. the auricular rate is 82·3, and the ventricular 24·7 per minute.

The question naturally arises whether there was, as we believe, complete auriculo-ventricular dissociation, or whether the ventricles responded not to each, but to every third or fourth, stimulus from the sinus. We know that such failure of the ventricles to respond to each stimulus from the sinus may be due to one or other of three causes—defective contractility of the ventricular musculature, depression of ventricular excitability, and lowered conductivity of the tissues of the auriculo-ventricular bundle. Further, we know that the conductivity of this bundle may be impaired either by an incomplete organic lesion of the bundle itself, or by negative dromotropic influence of vagus origin. Does any one of these three alternatives provide a wholly satis-

factory explanation of the facts recorded in the tracings, or are we forced to conclude that there was complete auriculo-ventricular heart-block due to an organic lesion of the *a-v* bundle?

After a most careful examination of all the tracings, we fail to detect any evidence of defective contractility on the part of the ventricular musculature. No indication of the alternating pulse was at any time observed. Nor do we find any proof that the infrequency of the ventricular beats and arterial pulse is the expression of a lowered excitability of the ventricular musculature. It is true that at the first glance the tracings in Plate I. suggest one of those rare and exceptional instances where the ventricles respond only to every third or fourth stimulus, and yet where the *a-c* interval is so short as to preclude the possibility of there being any defect in conductivity, and where we are consequently driven to suppose that the phenomena can only be due to a lowering of ventricular excitability. But a closer examination of the tracings, and particularly of those in Plates II. and III., shows how erroneous such an inference is. It is evident that we are not dealing with a 3 : 1 or 4 : 1 rhythm. On the contrary, the auricular and ventricular rhythms are wholly independent of each other, as is clearly demonstrated in the schematic diagrams accompanying the tracings.

We need, therefore, hardly consider in further detail the only other alternative explanation, namely, a partial auriculo-ventricular heart-block, with a 3 : 1 rhythm. It is sufficient to point to the tracings reproduced in Plates II. and III., which show the auricles to be beating rhythmically, and likewise the ventricles, but each with an independent rhythm and at a different rate, so that the ventricular systoles follow the auricular at varying intervals, sometimes preceding, at others coinciding with, the latter. The beating of the auricle and ventricle simultaneously is graphically recorded in the case of the fourth ventricular beat (pulse wave) and the eleventh auricular beat in Plate II., and of the seventh ventricular and twenty-first auricular beats in Plate III. In each instance the auricular and carotid waves coincide, and, for reasons which are sufficiently obvious and equally well known, there is in each instance one large wave $(a+c)$ in the jugular vein. As the tracings revealed a condition of complete, not partial, auriculo-ventricular heart-block, and as the condition persisted for some years, we concluded that, while negative dromotropic influence of vagus origin might well have been the exciting cause of the syncopal attacks in the earlier stages of the patient's illness before



Section, No. 143.

No. 179.

No. 203.

No. 229.

PLATE V.—Serial sections in the horizontal plane of the septum of a healthy human heart, illustrating the origin, position and course of the auriculo-ventricular bundle. The sections were treated with van Gieson's stain; the fibrous tissue consequently appears dark, the muscle tissue pale. (× 4.)

Section No. 245.

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Plate VI.—Further sections from the same heart as depicted in Plate V., to show the bifurcation of the auriculo-ventricular bundle and its two main branches. (× 4.)

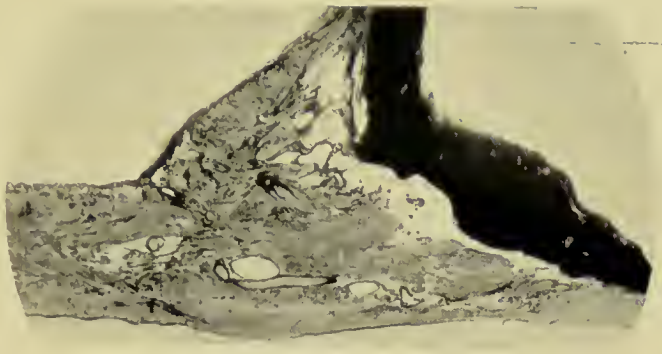

Fig. 1.

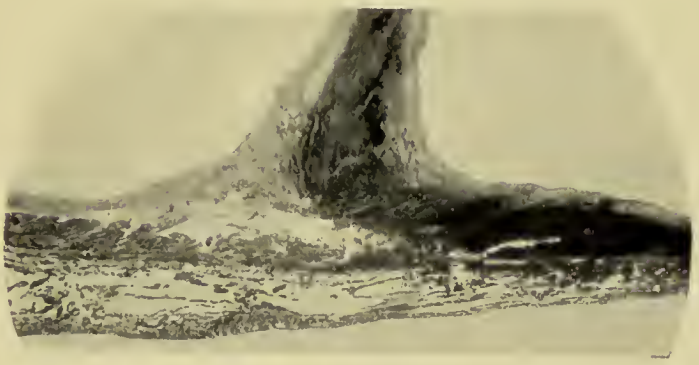

Fig. 2.

Fig. 3.

a-v bundle.

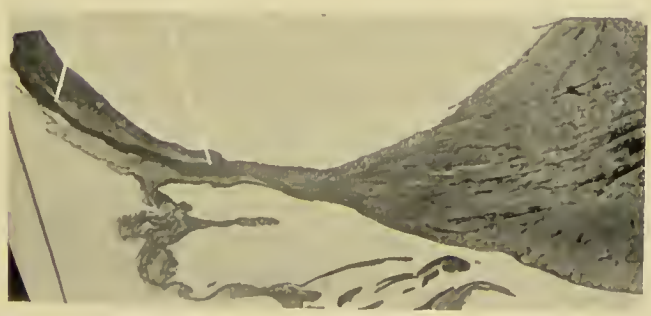

Fig. 4.

Plate VII. Sections of the septum of the heart described in the text. Figs. 1, 2 and 3 are horizontal sections taken from that portion of the septum which is indicated by the dotted line in Plate IV., Fig. 1. Fig. 4 shows a horizontal section passing through the upper part of the pars membranacea septi. (All × 4.)

the block became permanently complete, there was an organic lesion of the auriculo-ventricular bundle, leading eventually to complete failure on the part of the bundle to conduct the physiological stimuli to the ventricles.

As the tracings show complete auriculo-ventricular dissociation, it is to be expected that, just as the auricular and carotid waves sometimes coincide, so will the auricular and ventricular waves occasionally occur simultaneously, and we find it so. At times the ventricular wave appears quite distinct and apart from any other wave—for example, the ventricular wave between the eleventh and twelfth auricular waves in Plate II., or that between the twenty-first and twenty-second auricular waves in Plate III. But at other times the ventricular wave does coincide with an auricular one. The third auricular wave in Plate I. and the

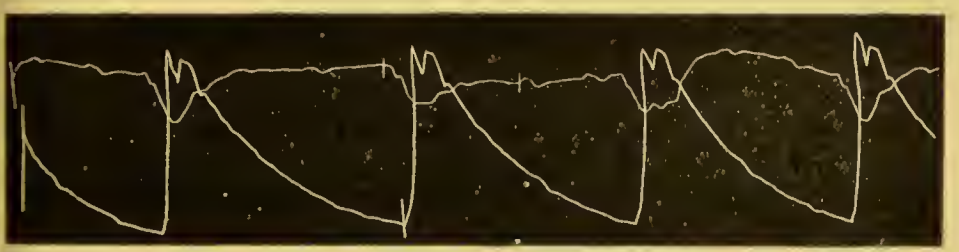

Fig. 3.—Tracings from the apex beat and radial pulse.

eighteenth auricular wave in Plate III. may be referred to in illustration of this fact.

As the occurrence of a ventricular wave indicates an antecedent contraction of the right ventricle, the tracings prove that the two ventricles beat synchronously, rhythmically, and at the same infrequent rate. The records of the movements of the left auricle are less clear than those of the right. We have elsewhere [1] shown records of heart-block in which large positive waves, produced by the systolic movements of the left auricle, appeared in the apical tracings. But even if we were unaware of the fact that when there is a condition of complete auriculo-ventricular heart-block the two auricles always continue to beat in unison, we should find, in Fig. 3, sufficient evidence that in the present instance the left auricle was beating at a rate and with a rhythm wholly independent of the left ventricle. In the upper tracing of Fig. 3, taken from the apex beat, there are a number of small

[1] *Proceedings of the Royal Society of Edinburgh*, 1905, vol. xxv. p. 1085; *British Medical Journal*, 1906, vol. ii. p. 1113.

positive waves appearing at a rate about three times as frequent as that of the left ventricle, as revealed by the systolic depression in the apical tracing or by the radial sphygmogram. Further, these small positive waves have a rhythm independent of that of the left ventricle, and thus they can owe their origin only to the beats of the left auricle. We have thus graphic evidence of the two auricles beating together at a rate of about 80 per minute, and likewise of the ventricles beating synchronously about 24 times per minute, and, further, of there being complete dissociation of the auriculo-ventricular rhythm.

The only other point in the tracings to which we would refer very briefly are the waves, indicated by the letter *s*, in the jugulo-carotid tracing of Plate III. We regard these waves, which must not be confounded with the *h* wave of Hirschfelder[1] and the *b* wave of A. G. Gibson,[2] as due to the increased pressure in the superior vena cava and jugular vein induced by the rhythmical contraction of the muscle fibres in the wall of the former vessel. This explanation of the wave we have already advanced in a former paper.[3] During the past two years our histological investigations have demonstrated that there is a considerable amount of striated cardiac muscle in the wall of the superior vena cava, extending as far up the vessel as the line of reflexion of the pericardium. We believe that under certain pathological conditions this muscle tissue may undergo such a degree of hypertrophy that its rhythmical contraction may be manifested, as in the case now recorded, by a positive wave preceding the auricular wave in graphic records taken from the jugular pulsations.

From 6th March 1903, when the last seizure, with one exception, occurred, until the end of his life, this state of complete heart-block persisted. In the spring of 1907 Sir William Gairdner resolved to leave Edinburgh and live in the neighbouring country, and about mid-summer he and his family entered upon their new house at Colinton. Shortly afterwards the end came with great suddenness, but was most characteristic in its peaceful serenity. On 28th June 1907 the annual dinner of the Royal Infirmary Residents' Club was held in Edinburgh, and amongst those present was Dr. Yellowlees of Glasgow. Early in the afternoon, Dr. Yellowlees, who had come from Glasgow in advance of the hour of dinner in order to call upon Sir William, spent some

[1] *Johns Hopkins Hospital Bulletin*, 1907, vol. xviii. p. 265.
[2] *The Lancet*, 1907, vol. ii. p. 1380.
[3] *The Practitioner*, 1907, vol. lxxviii. p. 589.

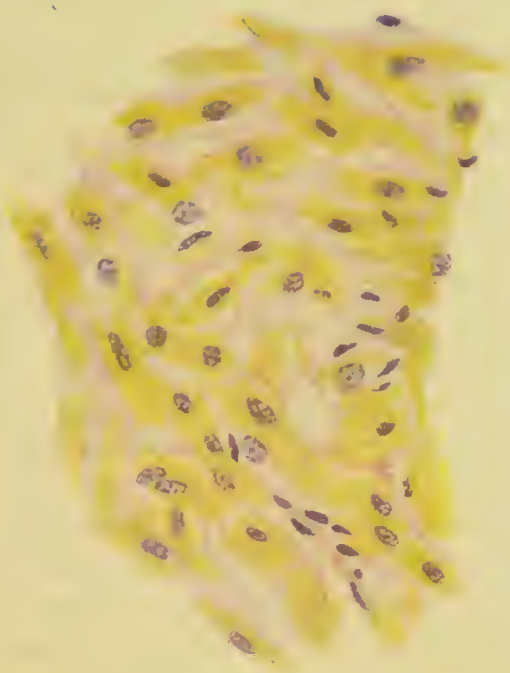

FIG. 1.—Portion of the auriculo-ventricular node in a healthy human heart. Hæmalum and van Gieson's stain. (× 350.)

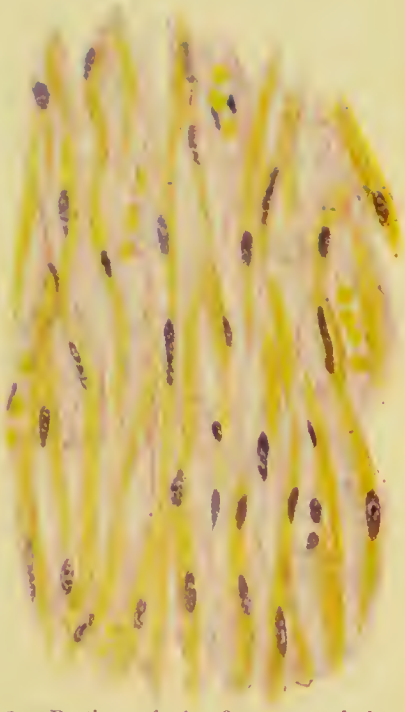

FIG. 2.—Portion of the first part of the auriculo-ventricular bundle in a healthy human heart. Hæmalum and van Gieson's stain. (× 350.)

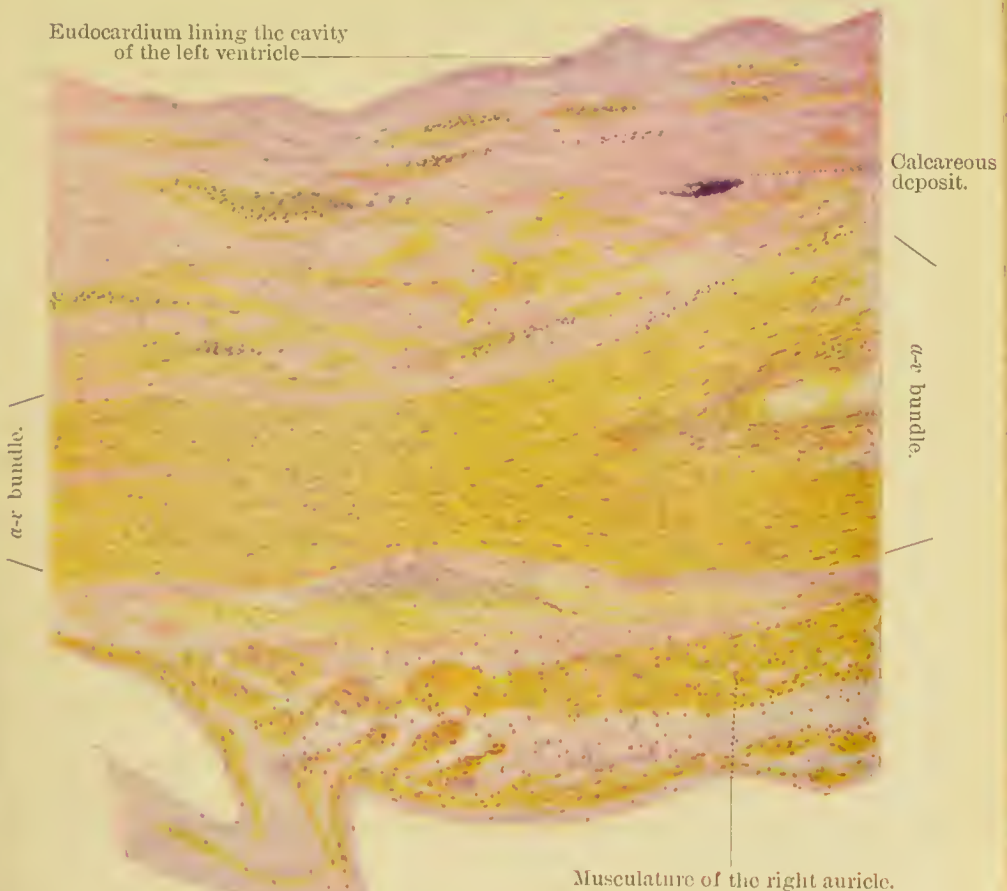

FIG. 3.—Section in a horizontal of plane the septum of the heart described in the text. The main stem of the *a-v* bundle is healthy. There are some small calcareous deposits in the fibrous tissue around it. Hæmalum and van Gieson's stain. (× 10.)

time with him at Colinton. Sir William had a long conversation with Dr. Yellowlees, and sent a friendly greeting by him to the Club; he afterwards accompanied him to the door, and then lay down upon his couch to read. One of the household looked in upon him, and at his request brought him another book. In less than an hour he was found to have passed away. In accordance with his desire a post-mortem examination was conducted the day following his death, and to the results of this we must now turn.

We do not propose to describe in any elaborate detail the condition of all the viscera as revealed at the examination, but we shall confine our observations mainly to the state of the heart, It will be sufficient if we note that the body was well nourished, and that no œdema or special degree of cyanosis was observed. The apices of the lungs presented small old fibrous cicatrices, and there were slight indications of arterio-sclerotic disease in the kidneys. The pericardium was perfectly healthy.

The heart was found to be greatly enlarged, the maximal external measurements of the organ being as follows:—

In the long axis	17 cm.
Transversely	12 cm.
Antero-posteriorly	11 cm
Circumference, 2 cm. below the auriculo-ventricular groove . . .	37·5 cm.
Surface of the ventricles along the line of the septum	25 cm.

The Right Auricle.—In the epicardium on the right side there were numerous yellow (fatty) streaks. Measured in the long axis of the heart the septal wall was 70 mm. long, whilst the lateral wall of the cavity was 105 mm. The orifices of the superior and inferior venæ cavæ, as well as those vessels themselves, were dilated. The coronary sinus was dilated; the valve of Thebesius guarding the orifice was well marked, thin, but not fenestrated. The endocardium lining the auricular cavity was smooth, but somewhat thickened. The fossa ovalis was closed. There was no other noteworthy feature regarding the septal wall of the auricle that could be recognised either by inspection or palpation. The tænia terminalis was hypertrophied, while the pectinate muscles were also hypertrophied. The dilatation of the auricle was manifested by the wide gaps between the pectinate muscles, allowing the endocardium and epicardium to come into contact with each other.

The Right Ventricle.—There was a moderate increase of the

epicardial fat over the right ventricle. The tricuspid orifice measured 14·5 cm. in circumference; the anterior and posterior papillary muscles were elongated and hypertrophied, and the chordæ tendineæ were slightly thickened but not shortened. The septal papillary muscles were represented by two small bundles, each 2 mm. in diameter, and some of the chordæ tendineæ passing to the septal cusp sprang directly from the septum. In the marginal cusp of the valve there were some irregular patches of fibrous thickening, but there was no calcareous change. The other two cusps were even less affected, being very little thickened and not calcareous.

The septal wall of the right ventricular cavity, measured from the auriculo-ventricular ring to the apex, was 90 mm., whilst the lateral wall measured 110 mm. The ventricular cavity was thus dilated. The columnæ carneæ were massive and thick. At the apex, however, the wall was not more than one-third of the normal thickness. The endocardium presented numerous whitish streaks and patches which, when examined microscopically, were found to be fibrous thickenings of the superficial layers of the endocardium. Some subendocardial fatty patches were also observed. These fibrous and fatty patches were all of small size, and were most abundant on the columnæ carneæ. The septum ventriculorum was carefully examined. Its muscular portion was hypertrophied throughout; the pars membranacea septi was large and uniformly thickened to a slight degree. No swelling, tumour, cicatrix, calcareous deposit or other local lesion was detected, on naked-eye examination, in any part of the septum.

The pulmonary orifice measured 9·8 cm. in circumference; the cusps were all healthy. The pulmonary vessel was somewhat dilated.

The Left Auricle.—There were four pulmonary veins, two on each side; none of them were more than slightly dilated. The muscular wall of the auricle was hypertrophied; the endocardium was appreciably thickened throughout. The auricular cavity was enlarged, measuring, along its lateral wall, 90 mm., and along its septal wall 70 mm., the measurements being taken in the long axis of the heart. The auricular appendix was distended at its commencement, but in its wall only the pectinate muscles were hypertrophied. There were no thrombi in the auricle or in its appendix.

The Left Ventricle.—The mitral orifice measured 11·5 cm. in circumference. The anterior, or aortic, cusp measured 3·5 cm. in

PLATE IX.

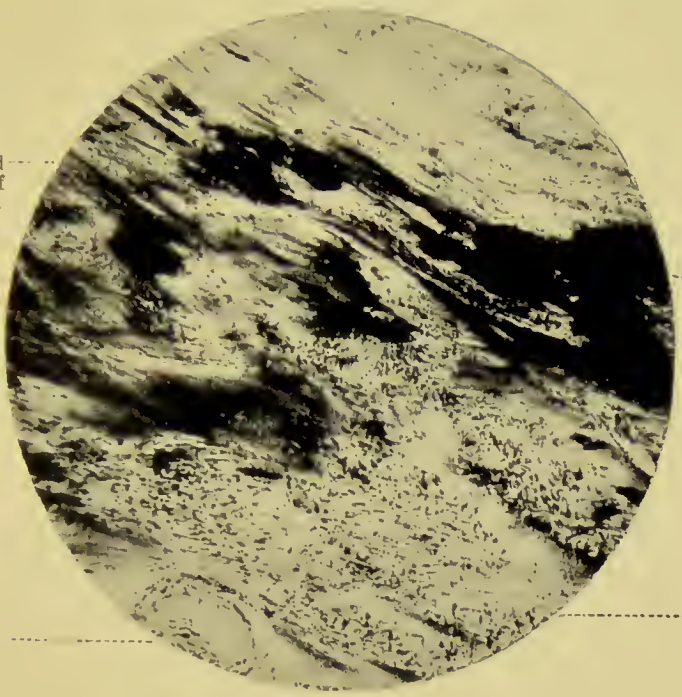

FIG. 1.—Heart-block. The junction of the auriculo-ventricular node and bundle, showing fibrosis and calcification. (× 50.)

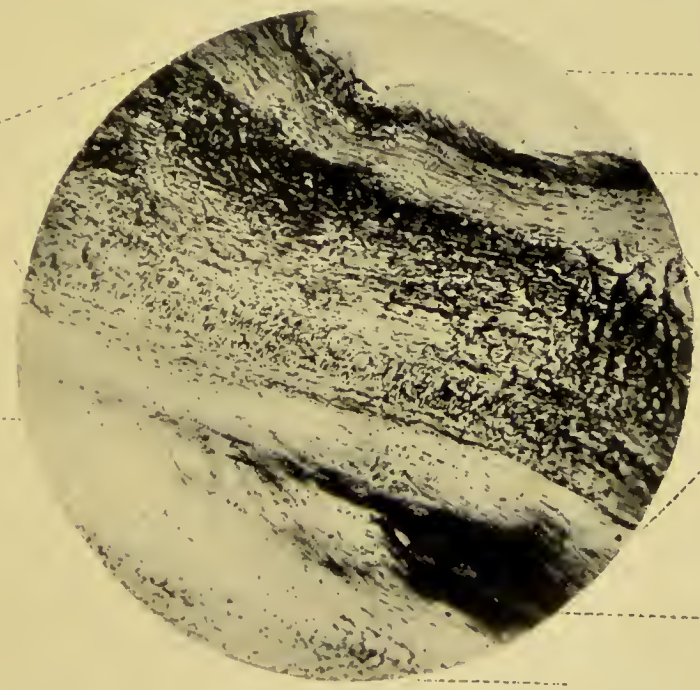

FIG. 2. Heart-block. The auriculo-ventricular bundle close to its point of bifurcation. In the fibrous tissue around the bundle there are some calcareous deposits, one of which is shown in the figure. (× 50.)

depth, and 5 cm. in width. It was thickened, particularly at its basal part, where it was also of yellowish colour, opaque, and firm. This increased thickness was derived in part from the attachments of the chordæ tendineæ radiating to the back of the valve. On microscopic examination of the basal part of the cusp, numerous fine calcareous deposits were detected. The posterior or marginal segment was also thickened. It was 6·5 cm. wide, and was never more than 1·7 cm. deep.

The chordæ tendineæ attached to the edge of the valve segments measured from 2·4 to 2·6 cm. in length, but those passing to the extreme tip of the papillary muscles averaged only 1·8 mm. in length. All the chordæ tendineæ were thickened, though not markedly so, but none were shortened. The fibrous change extended from the chordæ tendineæ into the tips of the papillary muscles. One of the chordæ passing from the posterior papillary muscle to the right margin of the posterior cusp presented, in its upper half, a club-shaped enlargement 4 mm. thick, which, when incised, was found to be composed of fibrous tissue. Several small pouches projected on the auricular surface of the posterior cusp. A small yellowish-brown opaque nodule, 1 mm. in diameter, which was attached to the auricular surface of the posterior cusp at the junction of its anterior and middle thirds and midway between its base and free margin, was evidently a partially organised vegetation. With this exception, there was no sign of recent endocarditis at any of the valvular orifices of the heart.

The mural endocardium presented patchy thickenings. On some of the columnæ carneæ these thickenings were represented by greyish-white fibrous areas lying with their long axes parallel to those of the columnæ carneæ. Several thin greyish-white fibrous cords passed between adjacent columnæ.

The length of the left ventricular cavity along its septal wall was 95 mm.; along its lateral wall 100 mm. The ventricular wall was, in general, hypertrophied. At the extreme apex the wall was, indeed, only 4 mm. thick, but at the level of the base of the anterior papillary muscle it was 1·7 cm. thick, and at a point 1·5 cm. below the auriculo-ventricular junction the thickness of the wall was 2 cm.

On the cut surface of the ventricular wall numerous pale greyish streaks and patches were observed, indicating fibrous transformation of the myocardium. These patches were most evident in the anterior papillary muscle (see Plate IV. Fig. 2) and in the myocardium in its vicinity. The branches of the coronary arteries which pass downwards and likewise those passing

inwards through the ventricular wall were conspicuous as greyish-white lines. Microscopic examination of portions of the muscular wall of the left ventricle showed that it was, for the most part, wonderfully healthy. There was but little fatty change in the muscle fibres. The fibrous transformation was not widespread, and was mainly confined to those situations in which it was apparent to the unaided eye.

The aortic orifice measured 9·2 cm. in circumference. The cusps and their lunulæ were all thickened, but the cusps were not adherent to one another, nor were they shrunken. The anterior cusp, in addition, presented a calcareous nodule at its base, extending into the cusp, but the calcification did not extend into the adjacent musculature. The left posterior cusp presented two small fibrous projections attached to the corpus Arantii, and in the left half of the cusp near its base there was a calcareous deposit similar to that in the anterior cusp. The right posterior cusp manifested a considerable degree of fibrous thickening over the corpus Arantii and also along the bases of the lunulæ, and in the posterior half of the basal part of this cusp there was some calcareous deposit. The sinuses of Valsalva were all dilated, the anterior and the left posterior sinuses being more markedly distended than the right posterior sinus.

The walls of both coronary arteries and of their branches were markedly atheromatous, with a considerable degree of calcification. These changes were clearly demonstrated by microscopic examination of the vessels and their branches.

The Aorta.—The ascending aorta, 2·5 cm. above the sinuses of Valsalva, measured 9·7 cm. in circumference. Its inner surface showed numerous, small, irregular, flat, yellow patches and streaks of fatty change in the superficial layers of the intima. Further, there were a few small patches of early atheroma. In the aortic arch, and especially where the great vessels are given off, there were several patches of well-marked atheroma. The deeper part of two of these patches consisted of soft pultaceous material, while in a third patch there was a calcareous plate. The intima of the aortic arch also presented several small patches of superficial fatty change.

The innominate artery was slightly dilated in a fusiform manner, and it showed considerable early atheromatous change. The carotid and subclavian arteries presented similar degenerative changes.

We are greatly indebted to Dr. Theodore Shennan for the kind assistance he gave us in examining the heart, and we gladly

express to him our grateful thanks for much invaluable help in obtaining the photo-micrographs which illustrate the salient features of the case.

From the foregoing description of the heart, it is apparent that the organ was hypertrophied; that the coronary arteries were the seat of atheroma; that the mitral and aortic valves were sclerosed and calcareous; and that there was some fibrous transformation of the ventricular musculature. It is further evident that, while on naked-eye examination of the heart the basal portion of the aortic cusp of the mitral valve was markedly indurated, no other gross lesion was detected, either in the auricular or the ventricular septum, which could be satisfactorily regarded as occasioning a loss of structural continuity of the auriculo-ventricular node or bundle.

It became necessary, therefore, to make a microscopic examination of this node and bundle, in order to ascertain whether there was any lesion of the primitive cardiac tissue in these situations, and if so, to determine its nature. At the same time, we proceeded to make a histological study of corresponding portions of healthy adult human hearts in order that we might have a clear conception, not only of the position of the auriculo-ventricular node, and the course, bifurcation and ramifications of the auriculo-ventricular bundle, but also of the microscopic characters of these portions of the primitive cardiac tissue. Four healthy hearts were examined. We removed such a portion of the cardiac septum that the block which was to be investigated contained the pars membranacea septi, the adjacent portions of the muscular septa—both ventricular and auricular—together with the central fibrous body, and the attachments of the septal cusp of the tricuspid valve and of the aortic cusp of the mitral valve. This portion of the cardiac septum was found to contain the auriculo-ventricular node, the bundle, and the first portions of its two main branches. In the case of the first healthy heart the block of tissue was cut in a plane which would be a frontal one were the long axis of the heart vertical. In the case of the remaining three healthy hearts, and also the heart with which we are more particularly concerned, the blocks of tissue were cut in serial sections from above downwards, and in the horizontal plane, supposing the long axis of the heart to be vertical. At the time when we commenced these investigations the only reliable and detailed information regarding the normal anatomy and histology of the auriculo-ventricular node and bundle was that

contained in the writings of Tawara[1] and of Keith and Flack.[2] So far as the human heart was concerned, however, Tawara's researches were confined to the fœtal heart and to that of children up to three years of age. When our examination of the five hearts was almost completed, Mönckeberg[3] published his monograph on the auriculo-ventricular bundle, this being the most extensive and elaborate contribution that has yet been made to the study of the bundle in the human heart.

Our own observations of the healthy node and bundle are in full agreement with the descriptions given by Tawara and Mönckeberg. In Plates V. and VI. there are reproduced certain of the serial sections of the third healthy heart that we examined. The sections were cut in the horizontal plane as already described, and were uniformly 0·03 mm. thick. They were stained with Mayer's hæmalum and van Gieson's stain, so that the pink fibrous tissue appears dark in the illustrations, whereas the muscle tissue, being stained yellow, appears paler. In the sections from No. 1 to No. 143 there is, as yet, no evidence of direct continuity between the auricular and the ventricular musculature. In section No. 179, however, we observe a tongue-like mass of vascular muscle tissue (the auriculo-ventricular node and the first part of the *a-v* bundle) projecting forwards from the musculature on the right side of the auricular septum, at a point opposite the attachment of the anterior mitral cusp to the central fibrous body of the heart, and somewhat behind the attachment of the septal cusp of the tricuspid valve. In section No. 203 the bundle is seen passing still further forwards, through the auriculo-ventricular septum, towards the pars membranacea septi, and at the same time passing somewhat to the left side. In other instances we find that the bundle does not pass over to the left, but takes up a central position in the septum, or remains constantly on the right side. In section No. 229, that portion of the bundle which is contained in the section is seen to lie for the most part on the left side of the septum, close to the endocardium. The bundle now contains numerous large blood-vessels. This section also shows that there is a thin branch passing backwards on the left side, sub-endocardially, and as if it were directed towards the insertion of the

[1] Tawara, *Das Reizleitungssystem des Säugetierherzens*, Jena, 1906.

[2] Keith and Flack, *The Lancet*, 1906, vol. ii. p. 359; *Journal of Anatomy and Physiology*, 1907, vol. xli., third series, vol. ii. p. 172.

[3] Mönckeberg, *Untersuchungen über das Atrioventrikulärbündel im menschlichen Herzen*, Jena, 1908.

aortic cusp of the mitral valve. This slender branch is described by Mönckeberg in the heart both of the fœtus and of the child. In section No. 245 (Plate VI.) we have now passed just below the deepest portion of the pars membranacea septi, and the bundle is seen to be dividing. Although the separation of the two branches is as yet incomplete, the left branch is spreading out in a fan-like fashion beneath the endocardium, on the left side of the septum. In section No. 247 the mass of ventricular musculature which is separating the two branches of the bundle from each other has become larger, but the two branches are still connected together both anteriorly and posteriorly. In section No. 253 the separation becomes complete, the left branch being spread out in the subendocardial tissue on the left side of the septum, while the right branch, presenting a somewhat crescentic form, lies on the right side. Section No. 259 passes well below the membranous septum, and the right and left branches still retain much the same relative positions as in section No. 253.

We did not trace the branches any further in this heart, but have done so in others. The left branch can be readily followed in serial sections. Its terminal ramifications, the Purkinje fibres, are most easily found in the subendocardial tissue in the neighbourhood of the anterior papillary muscle of the left ventricle. It is more difficult to follow the course of the right branch; and when it passes downwards, not in the subendocardial tissue but buried in the ventricular musculature, it may be, as Mönckeberg says, impossible to follow it, even in serial sections.

As regards the normal histology of the auriculo-ventricular node and bundle, we find our opinions are in all essential respects in agreement with the views expressed by Mönckeberg. The structure of the healthy auriculo-ventricular node in a young adult is depicted in Plate VIII. Fig. 1. The node consists of a highly irregular network of small muscle fibres, fusing one with another. The fibres present a faint transverse striation, and possess somewhat large ovoid nuclei, which stain somewhat less intensely than the smaller nuclei of the connective tissue cells. The muscle fibres are surrounded by thin connective tissue fibres, and small blood-vessels and capillaries are found in the meshes of the network. While ganglia and nerve fibres are readily enough detected in connection with the sino-auricular node of the hearts that we have examined, we have as yet failed to find any direct histological evidence of nerve tissue either in the auriculo-ventricular node or bundle. We do not mean to imply

that this node and bundle are devoid of nerve elements. The recent work of Gordon Wilson[1] is sufficient proof to the contrary. But our investigations indicate that nervous elements are certainly less abundant in the auriculo-ventricular node than in the sino-auricular node; and it is evident that special methods of examination are required to demonstrate their existence in the *a-v* node.

In structure the main stem of the healthy auriculo-ventricular bundle differs somewhat from that of the node whence it springs. The appearances are shown in Plate VIII. Fig. 2. The slender muscle fibres constitute a network; not a highly irregular one as in the node, but one in which the fibres fuse with one another at an acute angle. The meshes of the network are therefore much longer than they are broad, and the appearances are aptly compared by Mönckeberg to those of a collapsed fishing-net which is being pulled on at both ends. The muscle fibres show faint transverse striation; the nuclei are more spindle-shaped and stain more deeply than those of the node; and the muscle-fibres are encircled by fine strands of connective tissue. In the meshes of the network there are capillaries and small blood-vessels, the latter being in some instances larger and more abundant just behind the point of bifurcation of the bundle than elsewhere in its course. The transition between the histological characters of the auriculo-ventricular node and those of the bundle is a gradual one.

If we now proceed to consider the conditions which were found in Sir William Gairdner's heart, we may state at the outset that the only portions of the primitive cardiac tissue that was diseased were the auriculo-ventricular node and the first part of the auriculo-ventricular bundle. Both were markedly fibrous and calcified. In the lower portion of the bundle, in its branches and in the Purkinje fibres, we failed to detect any evidence of disease. We are fully aware that the amount of fibrous tissue in the node and bundle is usually greater in advanced life than in early manhood and childhood. But in the heart now under consideration the fibrous transformation was so marked and so intimately associated with calcareous deposits as to leave no doubt whatever that the appearances were not those of the aged healthy heart, but, on the contrary, were the direct result of pathological processes.

Two blocks of tissue were taken from the septum. The first

[1] *Proceedings of the Royal Society*, London, 1909, Series B. vol. lxxxi. No. B. 546, p. 151.

block, including the pars membranacea septi and portions of the auricular and ventricular septa around it, together with the anterior portion of the septal cusp of the tricuspid valve, was removed before the photograph reproduced in Plate IV. Fig. 1 was taken. The site and extent of the first block is therefore indicated in that figure by the black rectangular gap in the septum, partially covered by a portion of the infundibular cusp of the tricuspid valve which, when the photograph was taken, lay across the gap. This block was cut in serial section from above downwards and in the horizontal plane as already described. The block was found to contain the lower portion of the main stem of the bundle, its bifurcation, and the first part of its two branches. A second block of the auricular and ventricular septal tissues extending as far backwards as the mouth of the coronary sinus, and including the attachment of the aortic cusp of the mitral valve, was therefore removed. The size and extent of this block of tissue are indicated by the dotted rectangular area outlined in Plate IV. Fig. 1.

In the first block of tissue (see Plate VII. Fig. 4; Plate VIII. Fig. 3; and Plate IX. Fig. 2) we found no evidence of disease in the main stem of the bundle or in its branches. In Plate VIII. Fig. 3 we see a section of the septum stained by Mayer's hæmalum and van Gieson's stain. The main stem of the bundle is passing forwards from the right side of the auricular septum to assume a more central position. The bundle is enclosed in its connective tissue sheath, and in the latter there are some calcareous deposits which have taken the hæmatein stain. Further to the right of the bundle there is a portion of the auricular musculature, together with adipose tissue. In this illustration the bundle is seen to become broader as its passes forwards. This increase, however, is more apparent than real, being accounted for by the presence of a large blood-vessel which, as is shown in the illustration, is passing through the bundle somewhat obliquely to its long axis. Calcareous deposits were found in various parts of the fibrous sheath of the bundle. Such a deposit is seen in Plate IX. Fig. 2, which shows a portion of the main bundle somewhat further forward and further to the left than that in Plate VIII. Fig 3—close in fact to its point of bifurcation. A low-power view of the bundle, passing on the left side of the septum towards the thin pars membranacea septi, is also given in Plate VII. Fig. 4.

The striking pathological changes affected the auriculo-ventricular node and the first part of the bundle, as contained in the second block of septal tissue.

In the earlier sections from this block, which was cut in the horizontal plane, the auricular septum was found to contain much adipose tissue. The auricular muscle fibres were hypertrophied, but otherwise healthy. The aortic cusp of the mitral valve (see Plate VII. Fig. 1) was dense and fibrous, and contained numerous small calcareous deposits. In subsequent sections, passing lower down towards the line of attachment of the septal cusp of the tricuspid valve, the fibrous change spreads from the mitral valve into the auricular septum, and there is much calcareous deposit in the dense fibrous tissue. In Plate VII. Fig. 3 we observe how dense is the fibrous transformation. It is here that the auriculo-ventricular node and first part of the bundle are involved by the fibrosis and calcification. The condition is depicted in the photo-micrograph in Plate IX. Fig. 1, and in the coloured illustration in Plate X. Both the fibrosis and the calcification were extremely pronounced, and just as auriculo-ventricular dissociation may be induced by a complete section of the bundle or by the application of a clamp thereto, so, in our opinion, there is no doubt that the structural changes which we have described in the auriculo-ventricular bundle were amply sufficient to constitute an impassable barrier to the transmission of stimuli from auricle to ventricle.

The Purkinje fibres were found to be perfectly normal, and no evidence of disease was observed in the vagus nerves. The tissues at the junction of the superior vena cava and the right auricle presented no abnormality beyond the fact of there being a considerable increase of adipose tissue in this region.

Before concluding, we may refer very briefly to the conditions observed in other instances of heart-block. The earlier cases have already been discussed by us in a previous communication[1] in 1907. Since then, A. G. Gibson[2] has collected thirteen cases from the literature. He adds another which he himself examined; and in collaboration with Turrell[3] he has recorded yet another case. Lewis[4] collected records of twenty-seven cases, but after analysing them he concludes that in only seven was both the clinical and the histological evidence fully satisfactory. Nagayo[5] has still more recently analysed the literature of the subject.

[1] *The Practitioner*, 1907, vol. lxxviii. p. 589.
[2] *Quarterly Journal of Medicine*, 1908, vol. i. p. 182.
[3] *British Medical Journal*, 1908, vol. ii. p. 1486.
[4] *Ibid.*, 1908, vol. ii. p. 1798.
[5] *Zeitschrift für klinische Medizin*, 1909, Bd. lxvii. S. 495.

While a variety of lesions has been demonstrated, there are only three other cases, resembling that which we now record, where there is clear evidence of complete heart-block due to fibrous and calcareous changes in the auriculo-ventricular bundle. The three cases are those recorded by Beck and Stokes,[1] by Mönckeberg[2] (Heart No. CXIII.), and by Nagayo.[3]

[1] *Archives of Internal Medicine*, 1908, vol. ii. p. 277.

[2] *Untersuchungen über das Atrioventrikulärbündel im menschlichen Herzen*, Jena, 1908.

[3] *Zeitschrift für klinische Medizin*, 1909, Bd. lxvii. S. 495.